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# Metabolically healthy versus unhealthy obese phenotypes in relation to hypertension incidence; a prospective cohort study

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## Abstract

**Background:** Although obesity increases the risk of hypertension, the effect of obesity based on metabolic status on the incidence of hypertension is not known. This study aimed to determine the association between obesity phenotypes including metabolically unhealthy obesity (MUO) and metabolically healthy obesity (MHO) and the risk of hypertension incidence.

**Methods:** We conducted a prospective cohort study on 6747 adults aged 35–65 from Ravansar non-communicable diseases (RaNCD) study. Obesity was defined as body mass index above 30 kg/m<sup>2</sup> and metabolically unhealthy was considered at least two metabolic disorders based on the International Diabetes Federation criteria. Obesity phenotypes were categorized into four groups including MUO, MHO, metabolically unhealthy non obesity (MUNO), and metabolically healthy non obesity (MHNO). Cox proportional hazards regression models were applied to analyze associations with hypertension incidence.

**Results:** The MHO (HR: 1.37; 95% CI: 1.03–1.86) and MUO phenotypes (HR: 2.44; 95% CI: 1.81–3.29) were associated with higher hypertension risk compared to MHNO. In addition, MUNO phenotype was significantly associated with risk of hypertension incidence (HR: 1.65; 95% CI: 1.29–2.14).

**Conclusions:** Both metabolically healthy and unhealthy obesity increased the risk of hypertension incidence. However, the increase in metabolically unhealthy phenotype was higher.

**Keywords:** Metabolically unhealthy obesity, Metabolically healthy obesity, Hypertension, Incidence, PERSIAN

## Background

Hypertension is one of the strongest modifiable risk factors for cardiovascular disease (CVD) with its prevalence increasing especially in low- and middle-income countries [1, 2]. Reports indicate that a quarter of men and a fifth of women have hypertension, and hypertension is responsible for approximately 45% of deaths from CVDs

[3, 4]. Results of systematic review and meta-analysis on 42 Iranian studies showed that hypertension affects 22% of 402,282 subjects included in this analysis [5].

Many factors contribute to hypertension including sedentary lifestyle, kidney disease, diabetes, obesity, high salt intake, and processed foods [6, 7]. Among these factors, obesity contributes also to the development of CVDs, type 2 diabetes, cancer, and inflammatory diseases [8–11]. Evidence suggests that obesity, with its pro-inflammatory effects and oxidative stress, can cause insulin resistance, dyslipidemia, and other metabolic disorders. This is called metabolically unhealthy obesity (MUO)

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[12, 13]. Additionally, metabolically unhealthy non obesity (MUNO) phenotypes are at risk of type 2 diabetes, CVDs, fatty liver, and higher mortality [13, 14]. Nevertheless, some people with obesity have metabolically healthy status and they are said to have metabolically healthy obesity (MHO) phenotype [12]. Most studies focus on people with unhealthy metabolic status, and studies on MHO phenotype are limited. It is clear that MHO phenotype and its health consequences are not well understood [2, 15]. Evidence suggests that these people are at less risk for some of the mentioned diseases compared with MUO, but still have a higher risk of these diseases compared with people with normal weight. In general, MHO phenotype should not be considered a safe condition that does not require treatment for obesity [15].

Reports indicate that obesity is associated with the risk of developing hypertension. Since no study has examined the types of obesity phenotypes based on the metabolic status of individuals yet, the present study was conducted with the aim of identifying the association between metabolically healthy versus unhealthy obese phenotypes and the risk of hypertension incidence in the Ravansar non-communicable diseases (RaNCD) cohort study.

## Methods

### Study design and setting

We conducted a prospective cohort study using data from the RaNCD cohort study. The RaNCD study is the first cohort study on the Kurdish population in Iran which started in October 2014. The subjects are 35–65 years old and live in Ravansar city, Kermanshah province, Western-Iran. The RaNCD cohort study is a part of the PERSIAN (Prospective Epidemiological Research Studies in Iran) mega cohort study that was approved by the Ethics Committees in the Ministry of Health and Medical Education, the Digestive Diseases Research Institute, Tehran University of Medical Sciences, Iran. The details of this study were described in previous studies [16, 17]. In this study, we included all the recruitment phase participants who were surveyed from October 2014 to January 2017 and followed up until January 2021 ( $n=4764$  men and 5283 women). The RaNCD cohort study was approved by the Ethics Committee of Kermanshah University of Medical Sciences (No: IR.KUMS.REC.1400.268).

### Participants

Among RaNCD participants, 3300 were not included in the study for the following reasons: (1) Diseases such as CVDs ( $n=1709$ ), type 2 diabetes ( $n=870$ ), hypertension ( $n=1579$ ), cancer ( $n=83$ ), and thyroid diseases ( $n=763$ ); (2) pregnancy ( $n=138$ ); and (3) energy intake less than 800 kcal/day or more than 4200 kcal/day ( $n=737$ ). After excluding participants with missing data,

6747 participants were included into this study. Among them, 393 participants were identified as new cases of hypertension incident after follow-up, and the rest were considered as the sub-cohort group.

### Measurements

The current study obtained demographic data including age, sex, smoking status, and physical activity as well as medical history, medication, anthropometric indices, blood pressure, and biochemical analysis.

### Anthropometry

The height of all the participants was measured by the automatic stadiometer BSM 370 (Biospace Co., Seoul, Korea) with a precision of 0.1 cm in standing position without shoes. InBody 770 device (Inbody Co, Seoul, Korea) was applied to measure the weight and body fat mass (BFM) of participants with the least clothing and without shoes. To determine obesity, body mass index (BMI) was calculated by dividing the weight in kilograms by height in meters squared. BMI above  $30 \text{ kg/m}^2$  was considered as obesity. Waist circumference (WC) was measured using a non-stretched and flexible tape at the level of the iliac crest in standing position [18].

### Blood pressure

In RaNCD cohort study, conventional sphygmomanometry and auscultation of Korotkoff sounds was used to measure systolic and diastolic blood pressure (SBP and DBP) in sitting position after at least 4–5 min of rest. The blood pressure measurement was conducted twice with a 10 min interval and the average was calculated and reported as the final blood pressure [16].

### Biochemical analysis

25 cc blood samples were collected from all RaNCD participants. The serum and whole blood samples were subdivided and stored at  $-80^\circ\text{C}$  at the RaNCD cohort laboratory until analysis. Serum fasting blood sugar (FBS) was measured by glucose oxidase method. Total cholesterol (TC), high-density lipoprotein (HDL), triglyceride (TG) and low-density lipoprotein (LDL) concentrations were measured by enzymatic kits (Pars Azmun, Iran) [16].

### Obesity phenotypes

We defined MUO by the presence of  $\text{BMI} > 30 \text{ kg/m}^2$  and at least two metabolic disorders according to the International Diabetes Federation (IDF) statement [19] as follows:  $\text{HDL} < 40 \text{ mg/dl}$  in men and  $< 50 \text{ mg/dl}$  in women,  $\text{TG} > 150 \text{ mg/dl}$ ,  $\text{SBP} > 130 \text{ mmHg}$  or  $\text{DBP} > 80 \text{ mmHg}$  or receiving antihypertensive medication, and  $\text{FBS} > 100 \text{ mg/dl}$  or receiving medication for diabetes.

MHO was defined as BMI > 30 kg/m<sup>2</sup> and having at most one metabolic disorder mentioned above. MUNO phenotype was defined by the presence of BMI < 30 kg/m<sup>2</sup> and at least two of the above-mentioned metabolic disorders. Finally, MHNO participants were defined as healthy participants without obesity and metabolic disorder or having at most one metabolic disorder.

#### Outcome measurement of hypertension incidence

In the RaNCD study, participants are monitored for blood pressure each year and their systolic and diastolic blood pressure are measured. The medications and medical history of hypertension during the follow-up period are also assessed by the RaNCD physician. Hypertension was defined by SBP/DBP ≥ 140/90 mmHg and/or using anti-hypertensive medications in the time interval between baseline (first phase of Ravansar cohort which has been conducted since 2014) and hypertension diagnosis (from 2015 to 2021). The overall duration of the follow-up was 32,596 person/year.

#### Statistical analysis

Statistical analysis was performed using Stata, version 14 (Stata Corp, College Station, TX). Mean ± standard deviation (SD) and frequency percentage were used to report baseline characteristics of studied participants. To compare the results of baseline characteristics among different obesity phenotypes, one-way analysis of variance (ANOVA) was used for continuous variables, and

a Chi-square test was used for categorical variables. The number of degrees of freedom (df) used to calculate *P* values was 3.

Cox proportional hazards regression model was used to calculate hazard ratios (HRs) stratified by obesity phenotypes, with hypertension as the event and the time interval between baseline (first phase of RaNCD cohort) and hypertension diagnosis as the time covariate. This regression model was applied in the previous studies to determine hypertension incidence [20–23]. The model was adjusted for confounding variables including age, sex, physical activity, smoking and energy intake. HR was reported with 95% confidence interval (CI).

#### Results

A total of 6747 participants were analyzed in this study from which 6354 participants were part of the sub-cohort and the rest (n=393) were identified as new cases of hypertension. The percentage of hypertension new cases is demonstrated based on obesity phenotypes (Table 1).

The prevalence of MHO, MUNO, and MUO were 15.3, 17.4, and 8.03%; respectively. The mean physical activity in MHNO was significantly higher than the other three obesity phenotypes (MHO, MUNO, and MUO) in both men and women. Table 2 presents baseline characteristics of studied participants based on the obesity phenotypes.

The risk of incident hypertension increased in MHO phenotype compared to MHNO (HR: 1.41; 95% CI:

**Table 1** Baseline characteristics of studied participants

Variables	Total (n = 6747)	MHNO (n = 3965)	MHO (n = 1036)	MUNO (n = 1204)	MUO (n = 542)	<i>P</i> **
Age (year)	45.77 ± 7.76*	45.67 ± 7.97	44.99 ± 7.04	46.76 ± 7.88	45.86 ± 7.02	< 0.001
Weight (kg)	71.87 ± 13.42	66.49 ± 11.02	84.54 ± 10.83	71.76 ± 10.11	87.12 ± 12.25	< 0.001
WC (cm)	96.26 ± 10.36	92.03 ± 8.64	106.89 ± 8.23	95.94 ± 7.01	107.54 ± 8.81	< 0.001
BMI (kg/m <sup>2</sup> )	27.01 ± 4.67	24.73 ± 3.34	33.11 ± 2.93	26.45 ± 2.45	33.33 ± 3.28	< 0.001
BFM (kg)	24.27 ± 9.41	19.76 ± 6.61	36.38 ± 6.78	22.41 ± 5.25	36.24 ± 7.61	< 0.001
SBP (mmHg)	103.55 ± 12.42	101.62 ± 11.83	104.10 ± 11.31	107.16 ± 13.07	108.59 ± 13.83	< 0.001
DBP (mmHg)	67.44 ± 7.82	66.37 ± 7.36	67.76 ± 7.51	69.44 ± 8.28	70.18 ± 8.95	< 0.001
FBS (mg/dl)	89.92 ± 9.49	87.91 ± 8.03	88.96 ± 8.08	94.36 ± 11.29	96.58 ± 11.04	< 0.001
TC (mg/dl)	184.01 ± 36.79	180.03 ± 37.31	186.74 ± 33.72	190.40 ± 36.80	193.68 ± 34.36	< 0.001
TG (mg/dl)	130.01 ± 73.75	101.75 ± 46.35	114.06 ± 47.34	205.82 ± 87.54	198.37 ± 83.06	< 0.001
HDL (mg/dl)	46.83 ± 11.41	49.67 ± 11.21	49.82 ± 10.55	38.04 ± 7.45	39.85 ± 8.31	< 0.001
LDL (mg/dl)	101.26 ± 24.90	98.66 ± 25.16	102.64 ± 22.76	105.89 ± 25.49	107.33 ± 22.79	< 0.001
PA (MET hour/day)	41.08 ± 8.15	41.90 ± 8.78	39.71 ± 6.19	40.37 ± 7.78	39.32 ± 6.55	< 0.001
Current smoking (%)	11.9	20.7	9.4	23.1	14.1	< 0.001
Hypertension incidence	5.79	4.4	6.2	7.7	10.9	< 0.001

MHNO metabolically healthy non-obese, MHO metabolically healthy obese, MUNO metabolically unhealthy non-obese, MUO metabolically unhealthy obese, WC waist circumference, BMI body mass index, BFM body fat mass, SBP systolic blood pressure, DBP diastolic blood pressure, FBS fasting blood sugar, TC total cholesterol, TG triglyceride, HDL high density lipoprotein, LDL low density lipoprotein, PA physical activity

\*Mean ± SD

\*\**P* values were obtained one-way ANOVA and Chi square

**Table 2** Baseline characteristics of studied participants based on the different types of obesity phenotypes

Variables	Men (n = 3217)			Women (n = 3530)			P**		
	MHO (n = 2094)	MHO (n = 246)	MUNO (n = 692)	MUNO (n = 185)	MHNO (n = 1871)	MHO (n = 790)		MUNO (n = 512)	MUO (n = 357)
Age (year)	45.95 ± 7.88*	45.13 ± 7.09	45.96 ± 7.40	44.98 ± 7.01	45.35 ± 8.06	44.94 ± 7.03	47.84 ± 8.37	46.31 ± 6.99	<0.001
Weight (kg)	71.01 ± 10.74	94.50 ± 9.53	76.69 ± 8.93	95.74 ± 10.33	61.44 ± 8.94	81.44 ± 9.22	65.11 ± 7.47	82.65 ± 10.70	<0.001
WC (cm)	92.49 ± 8.30	107.53 ± 7.70	96.14 ± 6.87	106.97 ± 7.71	91.51 ± 8.97	106.68 ± 8.39	95.66 ± 7.20	107.83 ± 9.32	<0.001
BMI (kg/m <sup>2</sup> )	24.36 ± 3.40	32.42 ± 2.30	26.26 ± 2.52	32.37 ± 2.29	25.15 ± 3.22	33.32 ± 3.07	26.70 ± 2.34	33.82 ± 3.60	<0.001
BFM (kg)	17.74 ± 6.24	33.77 ± 6.69	20.91 ± 5.01	33.54 ± 7.17	22.31 ± 6.18	37.32 ± 6.58	24.71 ± 4.77	37.90 ± 7.41	<0.001
SBP (mmHg)	103.59 ± 11.53	108.20 ± 11.10	107.77 ± 12.22	110.03 ± 12.87	99.41 ± 11.77	102.83 ± 11.08	106.33 ± 14.11	107.84 ± 14.26	<0.001
DBP (mmHg)	67.31 ± 7.50	70.30 ± 7.65	69.98 ± 8.00	71.25 ± 8.77	65.32 ± 7.05	66.97 ± 7.29	68.72 ± 8.60	69.62 ± 9.00	<0.001
FBS (mg/dl)	88.32 ± 8.14	89.74 ± 8.57	93.71 ± 10.90	95.53 ± 9.94	87.45 ± 7.88	88.72 ± 7.91	95.25 ± 11.74	97.12 ± 11.55	<0.001
TC (mg/dl)	178.53 ± 36.02	187.56 ± 35.36	185.02 ± 33.58	188.54 ± 30.04	181.72 ± 38.64	186.49 ± 33.22	197.68 ± 39.64	196.34 ± 36.15	<0.001
TG (mg/dl)	108.41 ± 51.12	134.62 ± 67.99	216.65 ± 89.80	220.14 ± 86.05	94.28 ± 39.00	107.65 ± 36.49	191.19 ± 82.24	187.09 ± 79.27	<0.001
HDL (mg/dl)	46.37 ± 9.98	44.71 ± 8.98	35.24 ± 5.92	35.29 ± 5.74	53.39 ± 11.35	51.41 ± 10.50	41.82 ± 7.64	42.22 ± 8.46	<0.001
LDL (mg/dl)	99.23 ± 24.50	106.41 ± 23.50	103.03 ± 22.89	105.91 ± 20.73	98.03 ± 25.87	101.46 ± 22.42	109.77 ± 28.19	108.08 ± 23.78	<0.001
PA (MET hour/day)	43.70 ± 10.87	41.59 ± 9.87	41.31 ± 9.59	40.09 ± 9.78	39.89 ± 4.84	39.12 ± 4.30	39.10 ± 3.92	38.93 ± 3.91	<0.001
Current smoking (%)	35.9	30.7	34.8	35.3	3.7	2.8	7.1	3.1	0.001
Hypertension incidence	4	6.5	5.1	8.1	4.9	6.1	11.3	12.3	<0.001

MHNO metabolically healthy non-obese, MHO metabolically healthy obese, MUNO metabolically unhealthy non-obese, MUO metabolically unhealthy obese, WC waist circumference, BMI body mass index, BFM body fat mass, SBP systolic blood pressure, DBP diastolic blood pressure, FBS fasting blood sugar, TC total cholesterol, TG triglyceride, HDL high density lipoprotein, LDL low density lipoprotein, PA physical activity

\*Mean ± SD

\*\*P values were obtained one-way ANOVA and Chi square

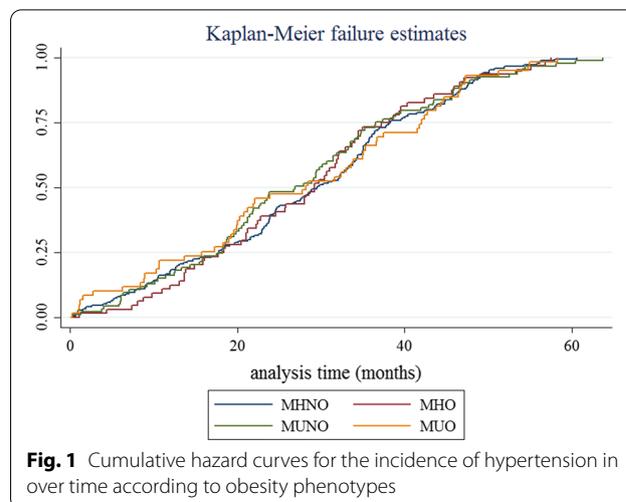
1.05–1.88) in model I, which remained significant after adjustment for age, sex, physical activity and smoking (HR: 1.37; 95% CI: 1.03–1.86). The risk of incident hypertension increased in MUO phenotype compared to MHNO (HR: 2.44; 95% CI: 1.81–3.29) after adjust sex and age, which remained significant after adjustment for age, sex, physical activity and smoking (HR: 2.40; 95% CI: 1.77, 3.26) (Table 3).

In addition, risk of hypertension significantly increased in MUNO phenotype compared to MHNO in all adjusted models (HR: 1.65; 95% CI: 1.29–2.14). The cumulative hazard curves show that the incidence of hypertension has increased by approximately 7% in MUO phenotype over 5 year; and this increase was more than other phenotypes over time (Fig. 1).

### Discussion

Our results show that both MHO and MUO phenotypes increase the risk of hypertension compared to MHNO phenotype. Furthermore, MUNO phenotype was associated with a higher risk of hypertension incidence compared to MHNO phenotype. Overall, the MUO phenotype increased the risk of hypertension incidence more than the other phenotypes in the follow-up time of the study. The obesity epidemic is growing and increases the risk of chronic non-communicable diseases leading to increased health system costs [24]. Epidemiological studies highlight the persistent link between obesity and hypertension, and the presence of obesity increases the risk of developing hypertension [7, 25, 26]. Since there are different phenotypes of obesity based on the metabolic status, we examined the association between obesity phenotypes and the risk of hypertension incidence.

The result of Whitehall II cohort study by Hinnouho et al. [27] on 5269 participants indicated that both obesity phenotypes, MHO and MUO lead to increased risk of mortality after seventeen years follow-up. Another prospective study by Fingeret et al. [28] did not find any difference between MHO and MUO in hypertension incidence after 10.9 years of follow-up (odds ratio (OR):



**Fig. 1** Cumulative hazard curves for the incidence of hypertension in over time according to obesity phenotypes

1.3, CI 95%: 0.8–2.09). Yuan et al. [29] showed that MHO has no association with the development of arterial stiffness (OR: 0.99; CI 95%: 0.61–1.6), while MUO and MUNO phenotypes lead to significantly progressed arterial stiffness (OR: 4.56; CI 95%: 2.60–8) and (OR: 5.05; CI 95%: 3.12–8.19), respectively. On the other hand, some studies by Hashimoto et al. [30] and Gilardini et al. [31] did not see any association between MHO and the risk of renal failure, prediabetes, diabetes and CVDs. In addition, Zhang et al. reported that none of the phenotypes were associated with an increased risk of left ventricular hypertrophy (MHO: OR: 0.845; CI 95%: 0.239–2.987; MUNO: OR: 0.567; CI 95%: 0.316–1.018; MUO: OR: 0.632; CI 95%: 0.342–1.166) [32]. Another study by Chaffin et al. [33] showed that MHO was not associated with incident CVD. In these studies, it has been interpreted that the reason for the lack of connection between MHO phenotype and the mentioned diseases is the favorable metabolic status. Furthermore, abdominal obesity has been considered more important in causing these chronic non-communicable diseases than overall obesity.

In the current study, we observed that MUO and MUNO increase the risk of hypertension incidence more

**Table 3** Hazard ratio of incident hypertension according to obesity phenotypes

Obesity phenotypes	N	% (N) of cases	Hazard ratio (95% CI)		
			Model I	Model II	Model III
MHNO	3965	4.4 (175)	Ref.	Ref.	Ref.
MHO	1036	6.2 (64)	1.41 (1.05, 1.88)	1.37 (1.02, 1.83)	1.37 (1.03, 1.86)
MUNO*	1204	7.7 (93)	1.68 (1.31, 2.16)	1.64 (1.27, 2.11)	1.65 (1.29, 2.14)
MUO	542	10.9 (59)	2.44 (1.81, 3.29)	2.36 (1.75, 3.20)	2.40 (1.77, 3.26)

Model I: adjusted for age and sex; Model II: adjusted for age, sex and physical activity; Model III: adjusted for age, sex, physical activity and smoking

MHNO metabolically healthy non-obese, MHO metabolically healthy obese, MUNO metabolically unhealthy non-obese, MUO metabolically unhealthy obese

than MHO. In addition, BFM and WC of the participants were higher in all three groups compared with the MHNO phenotype. Obesity, especially the excess visceral fat distribution, increases inflammatory cytokines and endothelial disorders in which several mechanisms contribute to hypertension [24, 34, 35]. Excess adipose tissue increases the production of pro-inflammatory factors such as leptin, tumor necrosis factor- $\alpha$ , interleukin-6, and resistin which contribute to the development of various metabolic diseases [36]. High calorie intake and increase in adipocytes stimulate  $\alpha$  and  $\beta$  adrenergic receptors, thereby increasing the activity of the sympathetic nervous system [37]. Obesity activates the renin-angiotensin system and the sympathetic nervous system, which leads to increased sodium reabsorption and arterial blood pressure [38, 39]. On the other hand, increasing adipose tissue leads to decreased adiponectin production and increased insulin resistance [40, 41]. Therefore, chronic hyperinsulinemia in obese people causes vasoconstriction and also increases urinary sodium reabsorption and is involved in the pathogenesis of hypertension [42]. In addition, increased circulating leptin levels in response to increased adipose tissue lead to impaired nitric oxide synthesis and ultimately vascular endothelial dysfunction [24]. In summary, the increased production of adipose tissue in obesity causes the production of pro-inflammatory cytokines which play an important role in the pathogenesis of hypertension by disrupting the metabolic status.

### Strengths and limitations

The present prospective study followed up the Kurdish population for the first time and examined the types of obesity based on metabolic status and the risk of hypertension incidence. In this study, we applied appropriate exclusion criteria, such as people who did not have normal calorie intake. However, this study had its limitations. First, the follow-up period seems to have been short. Second, the hypertension incidence was small for the study groups, and we could not assess the relationship between hypertension incidence and obesity phenotypes based on the sex, although it was adjusted for sex.

### Conclusion

In conclusion, the present study showed that both MHO and MUO phenotypes lead to an increase in hypertension incidence compared to MHNO phenotype. In addition, MUNO phenotype can also increase the hypertension incidence. However, MUO and MUNO phenotypes increase the risk of hypertension incidence more than MHO and MHNO phenotype. To prevent hypertension, maintaining normal body weight and

controlling central obesity as well as visceral fat is highly recommended.

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### Authors' contributions

FN, BH, SM and YP equally contributed to the conception and design of the research; FN, BH, ES and YP contributed to data collection; SM, YP and MD contributed to the acquisition and analysis of the data; SM, YP and MD contributed to the interpretation of the data; and SM, YP and MD contributed to draft the manuscript. SM, YP, MD, and NR contributed to revise the manuscript. All authors are in agreement with the manuscript and declare that the content has not been published elsewhere. All authors read and approved the final manuscript.

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### Availability of data and materials

Data will be available upon request from the corresponding author.

### Declarations

#### Ethics approval and consent to participate

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This study was approved by the Ethics Committee of Kermanshah University of Medical Sciences (ethics approval number: IR.KUMS.REC.1400.268). **Informed consent** Written informed consent was obtained from each studied subject after explaining the purpose of the study. The right of subjects to withdraw from the study at any time and subjects' information is reserved and will not be published.

#### Consent for publication

Not applicable.

#### Competing interests

Authors have no competing interests.

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